Central diabetes insipidus (DI) is a well-known complication of central nervous system trauma or tumors but is a rare complication of hypothermia. Review of the literature reveals scant case reports of DI as a complication of therapeutic hypothermia after cardiac pulmonary resuscitation or head injury, but to date there has been no mention of DI resulting from hypothermia alone.

**CASE**

A previously neurologically intact 13 year old Caucasian male with ADHD and ODD developed severe hypernatremia after developing profound hypothermia during treatment for antipsychotic induced toxic epidermal necrolysis. Despite aggressive interventions, external warming, his core temperature reached a nadir of 85.1°F (29.5°C) while his sodium concentration simultaneously increased. He required ECMO for temperature regulation and while his hypothermia improved, his hypernatremia worsened to 164 mEq/L despite fluid resuscitation. Initial septic work up for hypothermia was unsuccessful. Interestingly, within 48 hours of starting IV aqueous vasopressin for mild hypotension he became euhydrmic. However, when vasopressin was discontinued, his hypernatremia returned to 152 mEq/L with a serum osmolality of 332 mOsm/kg, an inappropriate urine osmolality of 400 mOsm/kg, and an inappropriately normal antidiuretic hormone level of 8.8 pg/ml (1.0-13.3). Efforts to discontinue vasopressin or DDAVP were unsuccessful as he would experience intermittent polyuria and hypernatremia. Pharmacologically, there were no medications that were reported to induce diabetes insipidus. He was subsequently presumed to have partial central diabetes insipidus and further pituitary workup revealed a normal Cortrosyn stimulation test and normal thyroid function tests. After he became more clinically stable, an MRI of the brain (c/s contrast) was obtained which showed an anatomically normal anterior pituitary and an appropriate location and presence of the posterior pituitary “bright spot.” Thereafter, trial off DDAVP was successful and he remained euhydrmic and euvoletic.

**CONCLUSIONS**

1. Hypothermia, either done therapeutically or as a consequence of an acute medical condition/ exposure, can be potentially complicated by diabetes insipidus which can then lead to increased morbidity and mortality if not promptly recognized.
2. Clinicians should consider and be aware of this potential sequela when performing therapeutic hypothermia and in patients who have undergone hypothermia and/or extreme temperature dysregulation from other conditions.
3. Unlike previous case reports citing hypothermia related DI, our case is not confounded by an incurring anoxic event. This unusual case encourages investigation into the role hypothermia may play in disrupting neuroendocrine functioning in pediatric patients.

**Figure 1:** On 2/17/14 patient noted to have spontaneous diuresis when briefly off DDAVP. This was associated with rebound hypernatremia to 157 and inappropriately low urine osmolality of 360 given serum osmolality of 327 at that time. Vasopressin was restarted for tachy pulse pressure control on 2/17/14 with normalization of sodium by 2/18/14.

**Figure 2:** A planned trial off DDAVP was undertaken on the evening of 3/10/14 with subsequent serial evaluations of sodium, urine osmolality and serum osmolality. Due to a marked rise in sodium with this intervention, DDAVP was restarted on 3/12 and patient return to euhydrmic.